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Search Results - Record(s) 1 through 2 of 2 returned.

☐ 1. Document ID: WO 200166522 A1, AU 200142451 A

Using default format because multiple data bases are involved.

L1: Entry 1 of 2 File: DWPI Sep 13, 2001

DERWENT-ACC-NO: 2001-596820
DERWENT-WEEK: 200204
COPYRIGHT 2005 DERWENT INFORMATION LTD
TITLE: New Benzoyl derivatives useful in controlling undesired growth of plant

INVENTOR: EDMUNDS, A; LUETHY, C ; SCHAETZER, J

PRIORITY-DATA: 2000CH-0000465 (March 9, 2000)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
WO 200166522 A1	September 13, 2001	E	064	C07D213/50
AU 200142451 A	September 17, 2001		000	C07D213/50

INT-CL (IPC): A01 N 43/40; A01 N 43/58; A01 N 47/02; A01 N 47/04; C07 C 311/07; C07 D 213/50; C07 D 265/02

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 2. Document ID: US 20030152921 A1, WO 200142451 A2, AU 200117265 A, US 20020102604 A1, EP 1252305 A2, JP 2003516150 W

L1: Entry 2 of 2 File: DWPI Aug 14, 2003

DERWENT-ACC-NO: 2001-367870
DERWENT-WEEK: 200355
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TITLE: Full length GENSET human nucleic acids encoding potentially secreted proteins, useful in gene therapy and vaccination against a variety of diseases, and for diagnosis of those diseases

INVENTOR: BOUGUELERET, L; DUMAS MILNE EDWARDS, J ; JOBERT, S ; MILNE EDWARDS, J D

PRIORITY-DATA: 2000US-187470P (March 6, 2000), 1999US-169629P (December 8, 1999), 2000US-0731872 (December 7, 2000), 2001US-0876997 (June 8, 2001)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
US 20030152921 A1	August 14, 2003		000	C12Q001/68
WO 200142451 A2	June 14, 2001	E	920	C12N015/09
AU 200117265 A	June 18, 2001		000	C12N015/09
US 20020102604 A1	August 1, 2002		000	G01N033/53
EP 1252305 A2	October 30, 2002	E	000	C12N015/09
JP 2003516150 W	May 13, 2003		000	C12N015/09

INT-CL (IPC): A01 K 67/027; C07 H 21/02; C07 H 21/04; C07 K 1/00; C07 K 14/00; C07 K 14/47; C07 K 16/18; C07 K 17/00; C12 N 1/15; C12 N 1/19; C12 N 1/21; C12 N 5/10; C12 N 9/00; C12 N 15/09; C12 P 21/02; C12 Q 1/68; G01 N 33/53

ABSTRACTED-PUB-NO: US20020102604A
BASIC-ABSTRACT:

NOVELTY - Full length GENSET human nucleic acids (I) encoding potentially secreted proteins (VI), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) an isolated polynucleotide (polyNt) (I) comprising a sequence encoding:

(a) a polypeptide (polyPt) comprising an amino acid (aa) sequence with at least 80% identity to at least 1 of 40 defined aa sequences ((A1)-(A40)) given in the specification, or at least 1 of the sequences of the polyPts encoded by clone inserts of the clone pool ATCC PTA-1218 (GENSET.071PRF); or

(b) a biologically active fragment of the polyPt;

(2) an expression vector (II) comprising (I);

(3) a host cell (III) recombinant for (I);

(4) a non-human transgenic animal (IV) comprising (III);

(5) a method (V) of making a GENSET polyPt, comprising:

(a) providing a population of host cells (III) comprising (I) operably linked to a promoter; and

(b) culturing the population of host cells (III) under conditions suitable for the production of the polyPt in the cell;

(6) a biologically active polyPt (VI) encoded by (I);

(7) an antibody (VII) that binds to (VI);

(8) a method (VIII) of determining whether a GENSET gene is expressed within a mammal, comprising:

(a) providing a biological sample from the mammal;

(b) contacting the sample with either:

(i) a polyNt that hybridizes under stringent conditions to (I); or

(ii) a polyPt that specifically binds to (VI);

(c) detecting the presence or absence of hybridization between the polyNt and an RNA species within the sample, or the presence or absence of binding of the polyPt to a protein within the sample (detection of hybridization or binding indicates that the GENSET gene is expressed within the mammal);

(9) a method (IX) of determining whether a mammal has an elevated or reduced level of GENSET expression, comprising:

(a) providing a biological sample from the mammal;

(b) comparing the amount of polyPt (VI), or an RNA species encoding it, within the sample with a level detected in or expected from a control sample (an increased/decreased amount of polyPt or the RNA species in the sample compared to the level detected in or expected from the control sample indicates that the mammal has an elevated/decreased (respectively) level of the GENSET gene expression); and

(10) a method (X) of identifying a candidate modulator of a GENSET polyPt, comprising:

(a) contacting the polyPt (VI) with a test compound; and

(b) determining whether the compound specifically binds to the polyPt (detecting specific binding between the compound and the polyPt indicates that the compound is a candidate modulator of the GENSET polyPt).

ACTIVITY - Variable.

MECHANISM OF ACTION - Gene therapy; vaccine.

No biological data given.

USE - (I) And the polyPt (VI) it encodes may be used in the prevention, treatment and diagnosis of diseases associated with inappropriate GENSET gene expression. For example, (I) (and vectors containing (I) (II)) and the encoded polyPt may be used to treat disorders associated with decreased GENSET gene expression by rectifying mutations or deletions in a patient's genome that affect the activity of GENSET by expressing inactive proteins or to supplement the patients own production of GENSET polyPts. Additionally, (I) and (II) may be used to produce the polyPts, according to standard recombinant DNA methodology (for example see Sambrook et al., Molecular Biology: A Laboratory Manual, (1989)), by inserting the nucleic acids into a host cell (III) and culturing the cell to express the protein (V) (the protein may be expressed either in vitro (e.g. in a fermentation culture) or in vivo (e.g. as part of a gene therapy procedure)).

Conversely, antisense/complementary nucleic acid molecules (I') may be administered to down regulate GENSET expression by binding with the cells own genes and preventing their expression.

(I) And (I') may also be used as DNA probes in diagnostic assays (e.g. polymerase chain reactions (PCR)) to detect and quantitate the presence of similar nucleic acid sequences in samples, and hence which patients may be in need of restorative therapy (i.e. method (VIII)).

They may also be used in the production of transgenic animals (IV) to study the expression and function of GENSET polyPts and their role in metabolism.

The GENSET polyPts (VI) may be used as antigens in the production of antibodies (VII) and in assays (i.e. method (X)) to identify modulators (agonists and antagonists) of GENSET polyPt expression and activity. The anti-GENSET antibodies and antagonists may also be used to down regulate expression and activity.

The antibodies may also be used as diagnostic agents for detecting the presence of GENSET polyPts in samples (e.g. by enzyme linked immunosorbant assay (ELISA), i.e. method (IX)) (claimed).

The diseases that may be prevented, diagnosed and treated using the methods vary depending upon the polyNt/polyPt sequences involved. Full details are given in the specification.
ABSTRACTED-PUB-NO:

WO 200142451A EQUIVALENT-ABSTRACTS:

NOVELTY - Full length GENSET human nucleic acids (I) encoding potentially secreted proteins (VI), are new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) an isolated polynucleotide (polyNt) (I) comprising a sequence encoding:

(a) a polypeptide (polyPt) comprising an amino acid (aa) sequence with at least 80% identity to at least 1 of 40 defined aa sequences ((A1)-(A40)) given in the specification, or at least 1 of the sequences of the polyPts encoded by clone inserts of the clone pool ATCC PTA-1218 (GENSET.071PRF); or

(b) a biologically active fragment of the polyPt;

(2) an expression vector (II) comprising (I);

(3) a host cell (III) recombinant for (I);

(4) a non-human transgenic animal (IV) comprising (III);

(5) a method (V) of making a GENSET polyPt, comprising:

(a) providing a population of host cells (III) comprising (I) operably linked to a promoter; and

(b) culturing the population of host cells (III) under conditions suitable for the production of the polyPt in the cell;

(6) a biologically active polyPt (VI) encoded by (I);

(7) an antibody (VII) that binds to (VI);

(8) a method (VIII) of determining whether a GENSET gene is expressed within a mammal, comprising:

(a) providing a biological sample from the mammal;

(b) contacting the sample with either:

(i) a polyNt that hybridizes under stringent conditions to (I); or

(ii) a polyPt that specifically binds to (VI);

(c) detecting the presence or absence of hybridization between the polyNt and an RNA species within the sample, or the presence or absence of binding of the polyPt to a protein within the sample (detection of hybridization or binding indicates that the GENSET gene is expressed within the mammal);

(9) a method (IX) of determining whether a mammal has an elevated or reduced level of GENSET expression, comprising:

(a) providing a biological sample from the mammal;

(b) comparing the amount of polyPt (VI), or an RNA species encoding it, within the sample with a level detected in or expected from a control sample (an increased/decreased amount of polyPt or the RNA species in the sample compared to the level detected in or expected from the control sample indicates that the mammal has an elevated/decreased (respectively) level of the GENSET gene expression); and

(10) a method (X) of identifying a candidate modulator of a GENSET polyPt, comprising:

(a) contacting the polyPt (VI) with a test compound; and

(b) determining whether the compound specifically binds to the polyPt (detecting specific binding between the compound and the polyPt indicates that the compound is a candidate modulator of the GENSET polyPt).

ACTIVITY - Variable.

MECHANISM OF ACTION - Gene therapy; vaccine.

No biological data given.

USE - (I) And the polyPt (VI) it encodes may be used in the prevention, treatment and diagnosis of diseases associated with inappropriate GENSET gene expression. For example, (I) (and vectors containing (I) (II)) and the encoded polyPt may be used to treat disorders associated with decreased GENSET gene expression by rectifying mutations or deletions in a patient's genome that affect the activity of GENSET by expressing inactive proteins or to supplement the patients own production of GENSET polyPts. Additionally, (I) and (II) may be used to produce the polyPts, according to standard recombinant DNA methodology (for example see Sambrook et al., Molecular Biology: A Laboratory Manual, (1989)), by inserting the nucleic acids into a host cell (III) and culturing the cell to express the protein (V) (the protein may be expressed either in vitro (e.g. in a fermentation culture) or in vivo (e.g. as part of a gene therapy procedure)).

Conversely, antisense/complementary nucleic acid molecules (I') may be administered to down regulate GENSET expression by binding with the cells own genes and preventing their expression.

(I) And (I') may also be used as DNA probes in diagnostic assays (e.g. polymerase chain reactions (PCR)) to detect and quantitate the presence of similar nucleic acid sequences in samples, and hence which patients may be in need of restorative therapy (i.e. method (VIII)).

They may also be used in the production of transgenic animals (IV) to study the expression and function of GENSET polyPts and their role in metabolism.

The GENSET polyPts (VI) may be used as antigens in the production of antibodies (VII) and in assays (i.e. method (X)) to identify modulators (agonists and antagonists) of GENSET polyPt expression and activity. The anti-GENSET antibodies and antagonists may also be used to down regulate expression and activity.

The antibodies may also be used as diagnostic agents for detecting the presence of GENSET polyPts in samples (e.g. by enzyme linked immunosorbant assay (ELISA), i.e. method (IX)) (claimed).

The diseases that may be prevented, diagnosed and treated using the methods vary depending upon the polyNt/polyPt sequences involved. Full details are given in the specification.

Terms	Documents
200142451	2

3937305 PMID: 9634557

Induction of caspase-3-like protease may mediate delayed neuronal death in the hippocampus after transient cerebral ischemia.

Chen J; Nagayama T; Jin K; Stetler R A; Zhu R L; Graham S H; Simon R P

Department of Neurology, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania 15213, USA.

Journal of neuroscience - the official journal of the Society for Neuroscience (UNITED STATES) Jul 1 **1998**, 18 (13) p4914-28,
ISSN 0270-6474 Journal Code: 8102140

Contract/Grant No.: NS 24728; NS; NINDS; NS 35965; NS; NINDS; NS 36736; NS; NINDS

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Delayed neuronal death after transient cerebral ischemia may be mediated, in part, by the induction of apoptosis-regulatory gene products. Caspase-3 is a newly characterized mammalian cysteine protease that promotes cell death during brain development, in neuronal cultures, and in other cell types under many different conditions. To determine whether caspase-3 serves to regulate neuronal death after cerebral ischemia, we have (1) cloned a cDNA **encoding** the rat brain **caspase-3**; (2) examined caspase-3 mRNA and protein expression in the brain using in situ hybridization, Northern and Western blot analyses, and double-labeled immunohistochemistry; (3) determined caspase-3-like activity in brain cell extracts; and (4) studied the effect of caspase-3 inhibition on cell survival and **DNA** fragmentation in the hippocampus in a rat model of transient global ischemia. At 8-72 hr after ischemia, caspase-3 mRNA and protein were induced in the hippocampus and caudate-putamen (CPu), accompanied by increased caspase-3-like protease activity. In the hippocampus, caspase-3 mRNA and protein were predominantly increased in degenerating CA1 pyramidal neurons. Proteolytic activation of the caspase-3 precursor was detected in hippocampus and CPu but not in cortex at 4-72 hr after ischemia. Double-label experiments detected **DNA** fragmentation in the majority of CA1 neurons and selective CPu neurons that overexpressed caspase-3. Furthermore, ventricular infusion of Z-DEVD-FMK, a caspase-3 inhibitor, decreased caspase-3 activity in the hippocampus and significantly reduced cell death and **DNA** fragmentation in the CA1 sector up to 7 d after ischemia. These data strongly suggest that caspase-3 activity contributes to delayed neuronal death after transient ischemia.

Jul 1 **1998**,

... 3 serves to regulate neuronal death after cerebral ischemia, we have (1) cloned a cDNA **encoding** the rat brain **caspase-3**; (2) examined caspase-3 mRNA and protein expression in the brain using in situ hybridization...

... cell extracts; and (4) studied the effect of caspase-3 inhibition on cell survival and **DNA** fragmentation in the hippocampus in a rat model of transient global ischemia. At 8-72...

... CPu but not in cortex at 4-72 hr after ischemia. Double-label experiments detected **DNA** fragmentation in the majority of CA1 neurons and selective CPu neurons that overexpressed caspase-3...

14304707 PMID: 10208968
Accumulation of caspase-3 messenger ribonucleic acid and induction of caspase activity in the ovine corpus luteum following prostaglandin F2alpha treatment in vivo.
Rueda B R; Hendry I R; Tilly J L; Hamernik D L
The Women's Research Institute, Wichita, Kansas 67214-3199, USA. brueda@kumc.edu
Biology of reproduction (UNITED STATES) May **1999**, 60 (5) p1087-92, ISSN 0006-3363 Journal Code: 0207224
Contract/Grant No.: R01-AG12279; AG; NIA; R01-HD34226; HD; NICHD
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
Caspase-3, a vertebrate homologue of the protein encoded by the Caenorhabditis elegans cell death gene, ced-3, induces apoptosis when overexpressed in eukaryotic cells. Since apoptosis occurs during corpus luteum (CL) regression in many species, including the ewe, these studies were conducted to 1) isolate a cDNA **encoding** ovine **caspase-3**, 2) measure steady state amounts of caspase-3 mRNA in the CL during luteolysis induced by prostaglandin F2alpha (PGF2alpha) and during the time of maternal recognition of pregnancy, and 3) measure changes in caspase activity during PGF2alpha-initiated luteal regression. Oligonucleotide primers corresponding to a human caspase-3 cDNA sequence were combined with total RNA from ovine CL in a reverse transcription-polymerase chain reaction-based procedure to amplify a 640-base pair partial cDNA with a nucleotide sequence 86% and 81% identical to the human and rat caspase-3 cDNAs, respectively. CL were collected from ewes at 0, 12, or 24 h after treatment with PGF2alpha on Day 10 of the estrous cycle and from nonpregnant and pregnant ewes on Day 12 or Day 14 of the cycle. Northern blot analysis of total cellular RNA from ovine CL and a radiolabeled ovine caspase-3 cRNA probe indicated the presence of a single mRNA transcript of approximately 2.5 kilobases. Levels of caspase-3 mRNA were approximately 3-fold higher ($p < 0.05$) in CL at 12 h and 24 h after PGF2alpha in comparison to those levels measured in matched CL from untreated ewes. There were no differences ($p > 0.05$) in amounts of caspase-3 mRNA in CL on Day 12 or Day 14 of the estrous cycle compared to Day 12 or Day 14 of pregnancy, respectively. Caspase activity in CL (measured by the ability of CL lysates to cleave an artificial caspase substrate) was also significantly ($p < 0.05$) increased in CL collected after treatment with PGF2alpha compared to CL collected from nontreated ewes. We conclude that physiological cell death during PGF2alpha-induced luteal regression in the ewe is mediated, at least in part, via increased expression and activity of the caspase family of pro-apoptotic proteases.

May **1999**,
...in many species, including the ewe, these studies were conducted to 1) isolate a cDNA **encoding** ovine **caspase-3** , 2) measure steady state amounts of caspase-3 mRNA in the CL during luteolysis induced ...
; Animals; Autoradiography; Blotting, Northern; Corpus Luteum--drug effects--DE; **DNA**--biosynthesis--BI; **DNA** --isolation and purification--IP; Enzyme Induction--drug effects--DE; RNA, Messenger --isolation and purification--IP...
Chemical Name: Enzyme Precursors; RNA, Messenger; Dinoprost; **DNA**; CPP32 protein; Caspases

6/3,K,AB/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

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      Set  Items  Description
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? s encod? (5n) (caspase (w)3)
         686996  ENCOD?
          54000  CASPASE
       10387084  3
      S1      55  ENCOD? (5N) (CASPASE (W)3)
? s encod? (5n) (caspase(w)7)
         686996  ENCOD?
          54000  CASPASE
        5961494  7
      S2       7  ENCOD? (5N) (CASPASE(W)7)
? s DNA or (nucleic) or polynucleotide
        2163744  DNA
         283872  NUCLEIC
          22872  POLYNUCLEOTIDE
      S3 2286522  DNA OR (NUCLEIC) OR POLYNUCLEOTIDE
? s (s1 or S2) and s3
            55  S1
             7  S2
       2286522  S3
      S4      43  (S1 OR S2) AND S3
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S5      23  RD (unique items)
? s s5 and py<2001
Processing
```

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? ds

Set     Items   Description
S1        154   (CASPASE(W)3) OR (CASPASE(W)7)
S2      19718   (DNA OR POLYNUCLEOTIDE)(5N) ENCOD?
S3         25   S1 AND S2
S4         43   SURVIVIN
S5          0   S3 AND S4
? s encod? (5n) (caspase(w)3)
          108879  ENCOD?
             481  CASPASE
         3643302  3
      S6       8  ENCOD? (5N) (CASPASE(W)3)
? t s6/3,k,ab/1-8

 6/3,K,AB/1
DIALOG(R)File 340:CLAIMS(R)/US Patent
(c) 2005 IFI/CLAIMS(R). All rts. reserv.

Dialog Acc No: 10338881  IFI Acc No: 2003-0083295
IFI Publication Control No: 2003-0083295  IFI Chemical Acc No: 2003-0022991
Document Type: C
ANTISENSE MODULATION OF CASPASE 3 EXPRESSION; AN OLIGONUCLEOTIDES ARE
HYBRIDIZABLE WITH NUCLEIC ACIDS TO INHIBIT THE GENE EXPRESSION OF ENZYME
Inventors: Cowsert Lex M (US); Zhang Hong (US)
Assignee: Unassigned Or Assigned To Individual
Assignee Code: 68000
Publication (No,Kind,Date), Applic (No,Date):
US 20030083295 A1 20030501 US 2002181107  20020711
Internat. Convention Pub(No,Date),Applic(No,Date):
 WO 2001US888   20010111
    Section 371:  20020711
    Section 102(e):20020711
Priority Applic(No,Date): US 2002181107   20020711

Abstract: Antisense compounds, compositions and methods are provided for
modulating the expression of caspase 3. The compositions comprise antisense
compounds, particularly antisense oligonucleotides, targeted to nucleic
acids encoding caspase 3. Methods of using these
compounds for modulation of caspase 3 expression and for treatment of
diseases associated with expression of caspase 3 are provided.


Abstract: ...of caspase 3. The compositions comprise antisense compounds,
particularly antisense oligonucleotides, targeted to nucleic acids
encoding caspase 3. Methods of using these compounds for
modulation of caspase 3 expression and for treatment of...

Exemplary Claim: ...An antisense compound 8 to 30 nucleobases in length
   targeted to a nucleic  acid molecule encoding caspase
   3, wherein said antisense compound specifically hybridizes with
   and inhibits the expression of caspase 3.
```

Induction of apoptosis in BPH stromal cells by adenoviral-mediated overexpression of caspase-7.
Marcelli M; Shao T C; Li X; Yin H; Marani M; Denner L; Teng B; Cunningham G R
Departments of Medicine, Baylor College of Medicine and VA Medical Center, Houston, Texas, USA.
Journal of urology (UNITED STATES) Aug **2000**, 164 (2) p518-25, ISSN 0022-5347 Journal Code: 0376374
Document type: Journal Article
Languages: ENGLISH
Main Citation Owner: NLM
Record type: Completed
PURPOSE: We hypothesized that expression/activity of critical components of the apoptotic pathway can be used to induce apoptosis of a human prostate cell line derived from benign prostatic hyperplasia (BPH) tissue. MATERIALS AND METHODS: We analyzed the apoptotic pathway in BPH cells treated with the powerful inducer of apoptosis, staurosporine (STS), and adenoviruses overexpressing caspase-3, -7, or the control gene lacZ. RESULTS: Twelve hours post-STS, most BPH cells were floating in the culture medium, TUNEL staining was widespread, and DEVDase activity (the catalytic activity of type II caspases) was increased. The pan-caspase inhibitor, Z-VAD-FMK, prevented STS-induced apoptosis. Based on these observations, we performed immunoblot analysis for the three known group II caspases (that is caspase-2, -3 and -7), but none of them was detected with three commercially available antibodies. Nevertheless, in view of the presence of increased DEVDase activity, we reasoned that a group II caspase must be a critical mediator of apoptosis in this model. If correct, we postulated that overexpression and activation of a type II caspase should cause apoptosis. To test this hypothesis, we coupled the cDNAs **encoding caspase-3** and **caspase-7** to adenoviral vectors and obtained constructs AvC3 and AvC7. Cells infected with AvC3 or AvC7 overexpressed the protein for caspase-3 or -7 within 24 to 48 hours. Caspase-3 overexpression did not cause apoptosis above that observed in cells receiving the control adenovirus expressing the lacZ cDNA (AvLac-Z). In contrast, caspase-7 overexpression induced massive apoptosis. BPH cells were then infected with increasing multiplicity of infection (MOI) of AvC7 and AvlacZ. A positive correlation was found between the amount of caspase-7 expressed and the level of DEVDase activity measured. AvC7 at MOIs of 25:1 and 50:1 induced apoptosis in about 50% of BPH cells at 72 hours post-infection. This effect was AvC7 specific, because the same MOIs of AvlacZ were not apoptogenic. CONCLUSIONS: Adenoviral-mediated overexpression of caspase-7 induces apoptosis of BPH-derived cells.
Aug **2000**,
... a type II caspase should cause apoptosis. To test this hypothesis, we coupled the cDNAs **encoding caspase-3** and **caspase-7** to adenoviral vectors and obtained constructs AvC3 and AvC7. Cells infected with AvC3 or AvC7...
; Apoptosis--drug effects--DE; Cells, Cultured; **DNA**, Complementary; Enzyme Inhibitors--pharmacology--PD; Genetic Vectors; Immunoblotting; In Situ Nick-End Labeling; Lac Operon...
Chemical Name: **DNA**, Complementary; Enzyme Inhibitors; Genetic Vectors; Ich-1 protein; Staurosporine; CPP32 protein; Caspases; caspase 7

6/3,K,AB/7 (Item 1 from file: 55)
DIALOG(R)File 55:Biosis Previews(R)

0012925252 BIOSIS NO.: 200100097091
Adenovirus-mediated gene transfer of p53-related p73 leads to apoptotic death of SK-N-As neuroblastoma cells
AUTHOR: Lo W D (Reprint); Zhu L; Akhmametyeva E; Chang L S
AUTHOR ADDRESS: Ohio State Univ, Columbus, OH, USA**USA
JOURNAL: Society for Neuroscience Abstracts 26 (1-2): pAbstract No.-302.6

2000 **2000**
MEDIUM: print
CONFERENCE/MEETING: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000; 20001104
SPONSOR: Society for Neuroscience
ISSN: 0190-5295
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The p73 gene was recently identified as a structural homologue of the p53 tumor suppressor gene. While the functional role is not known, the similarity to p53 suggests that p73 may participate in neuronal development and cell death. We tested whether p73 expression induces

hours, and transcript levels altered by ROCK inhibition were identified with oligonucleotide microarrays (GeneChips; Affymetrix, Santa Clara, CA). RESULTS: In these phenotypes, Y-27632 caused marked (twofold or more) increases or decreases in 14/4, 12/3, and 15/10 transcripts. In both fibroblast groups Y-27632-treatment increased expression of endothelin receptors and of parathyroid hormone-like hormone. The upregulation of alpha-smooth muscle actin in myofibroblasts was attenuated by Y-27632. Combining data from all groups identified ROCK-supported (Y-27632 inhibitable) expression of 10 transcripts, including ribonucleotide reductase M2, the **cyclin B1**-CDC2-**CKS2** system, and four mitotic spindle-associated proteins. CONCLUSIONS: ROCK inhibition causes broad inhibition of DNA synthesis and mitosis and causes changes that are different between (bFGF-activated) fibroblasts and (TGF-beta 1-induced) myofibroblasts. Thus, Rho/ROCK signaling regulates both common and distinct downstream events in corneal stromal cells activated (differentiated) to fibroblast or myofibroblast phenotype.

... identified ROCK-supported (Y-27632 inhibitable) expression of 10 transcripts, including ribonucleotide reductase M2, the **cyclin B1**-CDC2-**CKS2** system, and four mitotic spindle-associated proteins. CONCLUSIONS: ROCK inhibition causes broad inhibition of DNA...

4/3,K,AB/2 (Item 1 from file: 340)
DIALOG(R)File 340:CLAIMS(R)/US Patent

Dialog Acc No: 10668510
IFI Chemical Acc No: 2004-0050778
Document Type: C
REAGENTS AND METHODS FOR IDENTIFYING AND MODULATING EXPRESSION OF GENES REGULATED BY P21
Inventors: Chang Bey-Dih (US); Roninson Igor (US)
Assignee: Unassigned Or Assigned To Individual
Assignee Code: 68000
Publication (No,Kind,Date), Applic (No,Date):
US 20040175748 A1 20040909 US 2004801207 20040316
Priority Applic(No,Date): US 2004801207 20040316; US 99449589 19991129
Provisional Applic(No,Date): US 60-128676 19990409

Abstract: This invention provides methods and reagents for identifying genes involved in cell cycle progression, growth promotion, modulation of apoptosis, cellular senescence and aging, and methods for identifying compounds that inhibit or potentiate cellular senescence.

Non-exemplary Claims: ...5. The method of claim 4, wherein the gene is CDC2 (Acc. No. X05360), **CKsHs1** (CDC2 kinase) (Acc. No. X54941), PLK1 (polo-like kinase) (Acc. No. U01038), XCAP-H condensin...

...No. AA975298), EST (Acc. No. AA034414), EST (Acc. No. AA482549), Cyclin A1 (Acc. No. U66838), **Cyclin B1** (Acc. No. M25753), CDC25A (Acc. No. NM 001789), Dihydrofolate reductase (Acc. No. J00140), or ING1 ...

...13. The method of claim 12, wherein the gene is CDC2(Acc. No. X05360), **CKsHs1** (CDC2 kinase) (Acc. No. X54941), PLK1 (polo-like kinase) (Acc. No. U01038), XCAP-H condensin...

...No. AA975298), EST (Acc. No. AA034414), EST (Acc. No. AA482549), Cyclin A1 (Acc. No. U66838), **Cyclin B1** (Acc. No. M25753), CDC25A (Acc. No. NM 001789), Dihydrofolate reductase (Acc. No. J00140), or ING1 ...

...21. The method of claim 20, wherein the gene is CDC2 (Acc. No. X05360), **CKsHs1** (CDC2 kinase) (Acc. No. X54941), PLK1 (polo-like kinase) (Acc. No. U01038), XCAP-H condensin...

...No. AA975298), EST (Acc. No. AA034414), EST (Acc. No. AA482549), Cyclin A1 (Acc. No. U66838), **Cyclin B1** (Acc. No. M25753), CDC25A (Acc. No. NM 001789), Dihydrofolate reductase (Acc. No. J00140), or ING1 ...

4/3,K,AB/3 (Item 2 from file: 340)
DIALOG(R)File 340:CLAIMS(R)/US Patent

Dialog Acc No: 04034744
IFI Chemical Acc No: 2004-0008806
Document Type: C
REAGENTS AND METHODS FOR IDENTIFYING AND MODULATING EXPRESSION OF GENES REGULATED BY P21; INDUCING OVEREXPRESSION OF P21 IN MAMMALIAN CELL, ASSAYING CELL IN PRESENCE AND ABSENCE OF COMPOUND FOR CHANGES IN EXPRESSION OF SELECTED CELLULAR GENE WHOSE EXPRESSION IS MODULATED BY P21
Inventors: Chang Bey-Dih (US); Roninson Igor B (US)
Assignee: Illinois, University of
Assignee Code: 41184
Publication (No,Kind,Date), Applic (No,Date):
US 6706491 B1 20040316 US 99449589 19991129
Priority Applic(No,Date): US 99449589 19991129
Provisional Applic(No,Date): US 60-128676 19990409

Abstract: This invention provides methods and reagents for identifying genes involved in cell cycle progression, growth promotion, modulation of apoptosis, cellular senescence and aging, and methods for identifying compounds that inhibit or potentiate cellular senescence.

Exemplary Claim: ...whose expression is modulated by p21, wherein the cellular gene is CDC2 (Acc. No. X05360), **CKsHs1** (CDC2 kinase)(Acc. No. X54941), PLk1 (polo-like kinase) (Acc. No. U01038), XCAP-H condensin ...

...No. AA975298), EST (Acc. No. AA034414), EST (Acc. No. AA482549) Cyclin A1 (Acc. No. U66838), **Cyclin B1** (Acc. No. M25753), CDC25A (Acc. No. NM001789), Dihydrofolate reductase (Acc. No. J00140), or ING1 (Acc...

Non-exemplary Claims: ...is induced or repressed by p21, wherein the cellular gene is CDC2 (Acc. No. X05360), **CKsHs1** (CDC2 kinase) (Acc. No. X54941) PLK1 (polo-like kinase) (Acc. No. U01038) XCAP-H condensin...

...No. AA975298), EST (Acc. No. AA034414), EST (Acc. No. AA482549), Cyclin A1 (Acc. No. U66838), **Cyclin B1** (Acc. No. M25753), CDC25A (Acc. No. NM 001789), Dihydrofolate reductase (Acc. No. J00140), or ING1...
?

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? s cks or suc o
             1410  CKS
                0  SUC O
       S1    1410  CKS OR SUC O
? s cks? or suc
             1650  CKS?
             2461  SUC
       S2    4107  CKS? OR SUC
? s survivin
       S3    2550  SURVIVIN
? s s2 and s3
             4107  S2
             2550  S3
       S4       1  S2 AND S3
? t s4/3,k,ab/1

 4/3,K,AB/1     (Item 1 from file: 340)
DIALOG(R)File 340:CLAIMS(R)/US Patent
(c) 2005 IFI/CLAIMS(R). All rts. reserv.

Dialog Acc No: 10717159  IFI Acc No: 2004-0224408
IFI Publication Control No: 2004-0224408  IFI Chemical Acc No: 2004-0062331
Document Type: C
THAP PROTEINS AS NUCLEAR RECEPTORS FOR CHEMOKINES AND ROLES IN
TRANSCRIPTIONAL REGULATION, CELL PROLIFERATION AND CELL DIFFERENTIATION
Inventors: Amalric Francois (FR); Clouaire Thomas (FR); Girard
    Jean-Philippe (FR); Roussigne Myriam (FR)
Assignee: Unassigned Or Assigned To Individual
Assignee Code: 68000
Publication (No,Kind,Date), Applic (No,Date):
US 20040224408 A1 20041111 US 2003733878  20031210
Priority Applic(No,Date): US 2003733878   20031210
Provisional Applic(No,Date): US 60-432699    20021210; US 60-485027
20030703

Abstract: The invention relates to genes and proteins of the THAP family
comprising a THAP domain, and their use in diagnostics, treatment of
disease, and in the identification of molecules for the treatment of
disease. The invention also  relates to uses of THAP-type chemokine-binding
agents, such as THAP-family proteins, as a nuclear receptors for a
chemokines and to methods for the modulation (stimulation or inhibition) of
transcription, cell proliferation and cell differentiation as well as
methods for identifying for compounds which modulate THAP-chemokine
interactions.

Non-exemplary Claims: ...of claim 1, wherein said THAP responsive gene is
   selected from the group consisting of Survivin, PTTG1/Securin,
   PTTG2/Securin, PTTG3/Securin, CKS1, MAD2L1, USP16/Ubp-M, HMMR/
   RHAMM, KIAA0008/HURP, CDCA7/JPO1 and THAP1...
?
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      ---  -----  -----------
? s cks? or cks(2w)1 or cks(2w)2
Processing
Processing
            1650  CKS?
            1410  CKS
        12973740  1
              74  CKS(2W)1
            1410  CKS
        12174281  2
              23  CKS(2W)2
      S1    1650  CKS? OR CKS(2W)1 OR CKS(2W)2
? s cyclin(w)b1 or cyclin(w)B(w)1 or cyclinB1
Processing
           62165  CYCLIN
           63257  B1
            3031  CYCLIN(W)B1
           62165  CYCLIN
         3386235  B
        12973740  1
             151  CYCLIN(W)B(W)1
              73  CYCLINB1
      S2    3154  CYCLIN(W)B1 OR CYCLIN(W)B(W)1 OR CYCLINB1
? s s1 and s2
            1650  S1
            3154  S2
      S3       5  S1 AND S2
? rd
>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.
...completed examining records
      S4       3  RD (unique items)
? t s4/3,k,ab/1-3

 4/3,K,AB/1     (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
(c) format only 2005 The Dialog Corp. All rts. reserv.

16564704   PMID: 15223791
  Downstream effects of ROCK signaling in cultured human corneal stromal
cells: microarray analysis of gene expression.
  Harvey Stephen A K; Anderson Susan C; SundarRaj Nirmala
  Department of Ophthalmology, University of Pittsburgh, Pennsylvania
15213-2588, USA. harveysa@msx.upmc.edu
  Investigative ophthalmology & visual science (United States)  Jul 2004,
 45 (7) p2168-76, ISSN 0146-0404  Journal Code: 7703701
  Contract/Grant No.: EY03263; EY; NEI; EY05945-15S1; EY; NEI; EY08098; EY;
NEI
  Document type: Journal Article
  Languages: ENGLISH
  Main Citation Owner: NLM
  Record type: Completed
  PURPOSE: Rho-associated coiled-coil-containing protein kinase (ROCK) is a
downstream target of Rho GTPase signaling and regulates the assembly of
stress fibers. Previous reports indicate that Rho/ROCK signaling is
involved in the regulation of several cellular processes, some of which may
be cell-type specific and are probably critical to corneal stromal cell
activation. The present study identified ROCK-regulated gene expression in
corneal stromal cells. METHODS: Corneal stromal cells derived from eyes of
three different donors were cultured to yield the following designated
phenotypes: baseline fibroblasts (DMEM with 10% serum), activated
fibroblasts (10% serum+bFGF+heparin), and myofibroblasts (1% serum+TGF-beta
1). Cells were exposed to the ROCK inhibitor Y-27632 or vehicle for 12
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